

**"EVALUATION OF ISCHEMIC HEART DISEASE AND
EVALUATION OF ISCHEMIC HEART DISEASE AND
SYSTEMIC HYPERTENSION IN DIABETES MELLITUS**



Dissertation Submitted to
THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY
for
M.D. Degree in General Medicine
Branch I



March – 2008.

**DEPARTMENT OF GENERAL MEDICINE
COIMBATORE MEDICAL COLLEGE & HOSPITAL
COIMBATORE**

CERTIFICATE

This is to certify that the Dissertation entitled " Evaluation Of Ischemic Heart Disease and Systmic Hypertension in Diabetes Mellitus", herewith submitted by Dr.C. Suresh khanna, Post Graduate in General Medicine, Coimbatore Medical College to the Tamilnadu Dr. M.G.R. Medical University is a record of a bonafide research work carried out by him under my guidance and supervision from Jan 2006 to Jun 2007.

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DEAN

DECLARATION

*I solemnly declare that the Dissertation titled "**Evaluation Of Ischemic Heart Disease and Systemic Hypertension In Diabetes Mellitus**", was done by me at Coimbatore Medical College & Hospital during the period from Jan 2006 to Jun 2007 under the guidance and supervision of Prof. Dr. K. Umakanthan and Prof. Dr. Ramasamy.*

This dissertation is submitted to the Tamilnadu Dr. M.G.R. Medical University towards the partial fulfillment of the requirement for the award of M.D. Degree (Branch I) in General Medicine.

Place : Coimbatore
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ACKNOWLEDGEMENT

I sincerely thank Prof. Dr. A. KALANITHI, MD., Dr. HEMALATHA, MD., The Dean, Coimbatore Medical College Hospital, for having permitted me to use the hospital material for study.

I am extremely grateful to Prof. Dr. UMAKANTHAN, M.D., Professor and Head of the Department of Medicine for his expert help and friendly advice.

I am very grateful to Prof. Dr. THANDAPANI, M.D., D.M., Cardiology Chief, who had spent his precious time for doing Echo for all the patients I studied. I also extend my sincere thanks to Dr. BALA SUBRAMANIAN, M.D., D.M., Dr. DHARMARAJ, MD., DM Asst. Prof, of Cardiology for his valuable help and I also thank all the staff of Cardiology department.

I owe a great Debt of gratitude to Prof. Dr. RAMASAMY, M.D., Dr. S.PRABHA, M.D., for their generous and timely help in guiding and compiling the text and analysis of the reports in the course of preparation of this dissertation.

I extend my thanks to Dr. SHOBANA, M.D., Dr.S.USHA, M.D., Dr. RAVIKUMAR, M.D., Dr. MANSUR, M.D., Asst. professors in Medicine and my colleagues in giving me full support during this study. I also wish to thank the staff of the coimbatore Medical College Library for all help expended by them.

I would like to extend my heartfelt thanks to all the patients without whom this study would not have been possible.

CONTENTS

1.	INTRODUCTION	1
2.	AIM OF THE STUDY	5
3.	REVIEW OF LITERATURE	6
4.	MATERIALS AND METHODS	42
5.	DATA ANALYSIS OF RESULTS	46
6.	DISCUSSION	53
7.	CONCLUSION	58
8.	BIBLIOGRAPHY	59
9.	APPENDIX	66

PROFORMA

MASTER CHART

“EVALUATION OF ISCHEMIC HEART DISEASE AND SYSTEMIC HYPERTENSION IN DIABETES MELLITUS”

INTRODUCTION

DEFINITION

Diabetes mellitus is a metabolic disorder, due to relative or absolute lack of insulin secretion and or action, resulting in elevated blood glucose level with long term vascular and neurological complications¹. With better control of metabolic and infective complications, diabetes has predominantly become a disease of cardiovascular system².

EPIDEMIOLOGY

Diabetes Mellitus is world wide in distribution and varies between different populations. The prevalence of Type₁ Diabetes Mellitus is highest in Scandinavia and lowest in Japan. The American National Diabetes Data group utilising 75 Gm Oral Glucose has estimated the prevalence of diabetes Mellitus - 6.6% and Impaired GTT -11.2%.

CLASSIFICATION OF DIABETES MELLITUS⁴

- 1.Type₁ Diabetes Mellitus
- 2.Type₂ Diabetes Mellitus
- 3.Malnutrition Related Diabetes Mellitus (MRDM).
- 4.Other types associated with certain conditions and syndromes :
 - ✓ Pancreatic abscess, disease of hormonal aetiology, drug or chemical induced conditions.
 - ✓ Abnormalities of insulin or its receptors, certain genetic syndromes and miscellaneous.
5. Impaired glucose Tolerance (Obese, Non-Obese).
6. Gestational Diabetes Mellitus.

DIAGNOSIS⁵

Fasting Plasma Glucose above 126mg% or Random glucose more than 200mg% on more than one occasion, the diabetes can safely be made.

SYMPTOMS OF DIABETES MELLITUS

Classical symptoms of diabetes are 'Polys' - (Polydipsia, Polyuria /Polyphagia). The other symptoms are weight loss, asthenia, pruritus vulvae or balanitis or presents with macro or microvascular complications. Myocardial ischemia or infarction may be silent, because of blunted appreciation of pain and most often atypical symptoms such as confusion, dyspnoea, fatiguability or nausea or resting tachycardia. Most of the patients are asymptomatic and found to be diabetic on routine examination.

DIABETES AND CARDIOVASCULAR DISEASE⁶

The morbidity and mortality in diabetes is attributed predominantly to diabetic heart disease⁷. Coronary Artery Disease (CAD) is about twice as frequent in diabetic women compared to respective non-diabetics. One third of all deaths in DM patients after forty years of age has been attributed to CAD.

CAD in diabetic patients is characterised by greater prevalence of triple vessel disease and diffuse⁸ coronary atherosclerosis as evidenced by post mortum study and histological analysis. Linear arterial calcification has been found to correlate with the degree of glucose intolerance. In autopsies conducted in Type₁ Diabetes Mellitus patients, the lumen of coronary vessel was reduced by half in about 50% of examined arteries compared to 1% in non-diabetics.

In fact 75% subjects sustaining acute myocardial infarction before 45 years of age have some form of glucose intolerance. systemic hypertension acting in concert with Diabetes Mellitus results in significant mortality and morbidity⁹. Blood Pressure has been found to be 10-12 mm higher in diabetic subjects compared to the general population. Prevalence of hypertension is about 40-60% in Type₂ Diabetes Mellitus patients¹⁰.

The scenario of hypertension is different in Type₁ Diabetes Mellitus and Type₂ Diabetes Mellitus. Hypertension in Type₁ Diabetes Mellitus often appears with onset of nephropathy while in Type₂ Diabetes Mellitus 50% of patients are hypertensive at the time of diagnosis.

Aim of the Study

AIM OF THE STUDY

1. To study the incidence of both Ischemic Heart Disease and Systemic Hypertension in Diabetes Mellitus.
2. Evaluating the age and sex incidence of Systemic Hypertension and Ischemic Heart Disease in Diabetes Mellitus.
3. Evaluating the risk factors involved in causation of cardiovascular complications.
4. To study the prevalence of Silent Myocardial Infarction and Ischemia in DM patients.
5. To identify the maximum duration taken for the development of cardiovascular complications in DM patients.

Review of Literature

REVIEW OF LITERATURE

DEVELOPMENT AND CONCEPT OF CARDIO-DIABETOLOGY¹¹

With better control of metabolic and infective complications, diabetes has predominantly become a disease of the cardiovascular system. Cardio-Diabetology is emerging as a sub-speciality throughout the globe to tackle the menace of cardiac related mortality in diabetes, particularly Type₂ Diabetes Mellitus

Cardiac involvement in diabetes mellitus commonly manifests as CAD, which alone accounts for 40% of deaths in diabetes during 40's and this mounts to 50% in the sixth decade of life. About the age of 65 years, 70% of diabetic patients of either sex die of cardiac involvement.

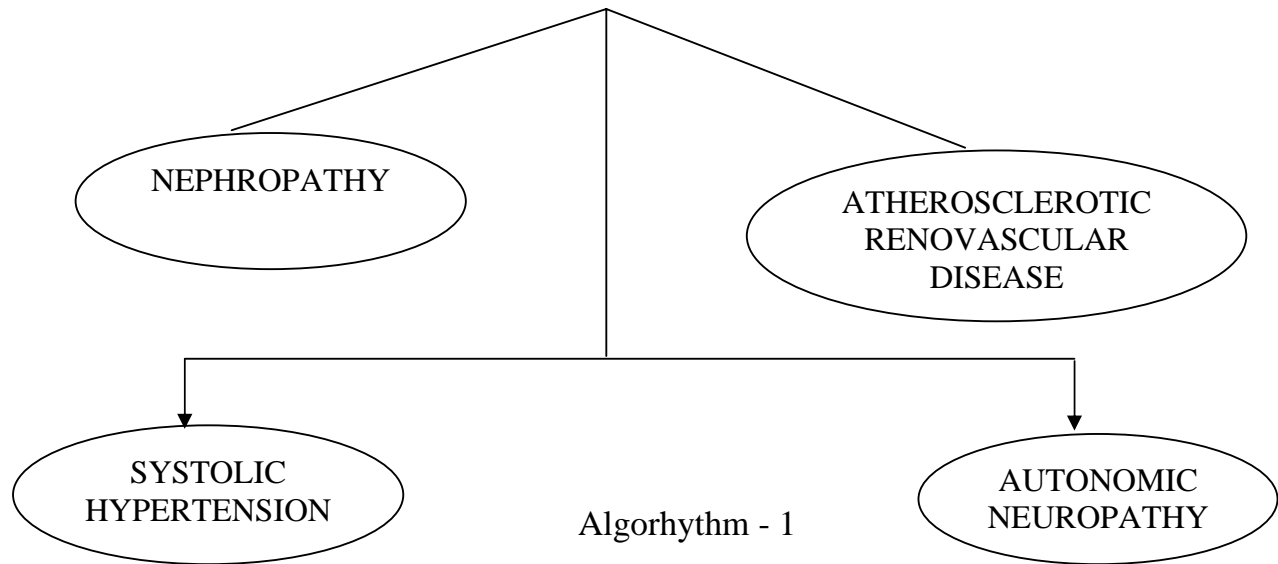
CAD is the leading cause of mortality in Diabetes. It not only involves the epicardial coronary arteries but also the intramural coronary arteries. Silent ischaemic events constitute a special feature of presentation. CAD in DM patients is not only frequent but occurs at younger age and the involvement is more extensive. Triple vessel disease and the left main stem lesion are more common.

Sudden cardiac death is the most dreadful complication and it is 1-8 times more common in men and 3 times common in women compared to non-diabetics. CAD, Dilated cardiomyopathy and autonomic neuropathy contribute to this morbidity and mortality.

Systemic Hypertension in diabetic patients is not un-common. The data from National Institute of Health USA shows that incidence of Hypertension is 2.8 times higher among diabetes below 45 years of age compared to the non-diabetics. Data from India is scanty and reveal varying prevalence of hypertension among diabetic patients.

Recognition of Hypertension in Diabetes Mellitus is of crucial importance because it is a major risk factor for development of complications in Diabetic patients¹². It is also a great risk factor for the development of macrovascular disease. Diabetes Mellitus patients are more prone to develop coronary Heart disease especially when there is associated left ventricular Hypertrophy¹³. (Algorhythm1).

DIABETES RELATED HT^{14,15}



IN UTERO EVIDENCE¹⁶

Low birth weight (Less than 2.5 Kg) is likely to predispose to the development of impaired glucose tolerance and Type₂ Diabetes Mellitus at a later age. The low birth weight internalises adaptive metabolic process in-utero which, if subsequently followed by normal or abundant supply of food during childhood, adolescence and adult years, result in the development of IGT, Type₂ Diabetes Mellitus, Hypertension and possibly dyslipidemias and coronary artery disease in the middle years of life. This was earlier called '*Double - Trouble-Syndrome*'. These evidences were generated through the studies in U-K, Sweden, USA and some other parts of the world. Hertfordshire-Cohort study reveals that men with lowest birth weight at birth and at one year had the highest death rates from Ischemic Heart Disease.

PATHOGENESIS OF CORONARY ARTERIAL HEART DISEASE IN DIABETES MELLITUS¹⁸

I.IMPACT OF HYPERGLYCAEMIA AND HYPERINSULINEMIA

Coronary heart disease occurs 3 times greater in frequency in patients with Type₂ Diabetes Mellitus. Females lose their immunity against CHD when they have Type₂ Diabetes Mellitus. Accelerated atherosclerosis causing macrovascular diseases affecting coronary, cerebral or peripheral arteries, is the main cause of morbidity in patients with Diabetes Mellitus. The endothelial cells of the vessels bears the brunt of various pathogenic process leading on to vascular occlusion¹⁹ as shown in the table no:1.

S.No	Particulars
1.	Endothelial injury with resultant increased permeability.
2.	Migration of various cells into the sub endothelial tissue
3.	Adherence of platelets to subendothelial collagen
4.	Migration of macrophages
5.	Smooth muscle cell proliferation and plaque formation
6.	Platelet aggregation leading to thrombogenesis
7.	Vascular occlusion

CASCADE OF EVENTS LEADING TO ENDOTHELIAL INJURY

TABLE NO: 1

Hyperglycaemia is the hall mark of Diabetes Mellitus. It is responsible for all the complications in DM Patients. It leads on to the following biochemical changes.

a) GLYCATION

The non-enzymatic linkage between a reducing sugar and a receptive aminoacid, indeed widespread, affecting virtually every tissue protein. The most relevant proteins which involved in atherosclerosis are.

1. Lipoproteins²⁰

Of the elevated lipoproteins such as total cholesterol, Triglycerides, low density lipoprotein (LDL) and very low density lipoprotein, glycation of LDL is seen in 3 or 4 times higher in diabetic patients. Apolipoproteins A1, A11, B, C & E are also glycated to a greater extent²¹. LDL is deposited in the blood vessel wall which undergo modification and degradation to form insoluble complexes are referred as Advanced Glycation End products (AGE) with potential pathophysiological consequences. Normally, LDL is metabolised after recognition by the LDL receptors, but due to glycation of its lysine residue it interferes with its recognition by LDL receptor and thus has to go through the alternative pathway of endocytosis by macrophages - thus forming foam cells which is characteristic of the early atherosclerotic plaque and LDL & LP (a) plays a major role in atherosclerosis²².

2. Coagulation proteins

Glycation of coagulation proteins particularly antithrombin III and fibrin results in interference of the cleavage of fibrinogen.

3. Structural proteins

Collagen - most abundant proteins in the body which is rich in lysine, and most vulnerable to AGE formation due to glycation. The AGE formed on long lived collagen in the walls of blood vessels can trap LDL which will undergo AGE formation due to glycation results in atherosclerosis.

b) Hyperglycemia and Intracellular Hypoxia²³

Hyperglycemia induces intracellular hypoxia due to increase in oxygen demand by the altered metabolic substrates and glycohemoglobin has greater affinity for oxygen. Hyperglycemia has its impact on 2-3 DPG in RBC which leads to tissue hypoxia. There is a rise in free cytosolic NADH / NAD ratio called *Pseudohypoxia*. This cellular hypoxia produces microangiopathy which may be responsible for damage to the media of large size arteries.

HYPERINSULINEMIA²⁴

Excessive concentration of insulin might contribute to the development of atherosclerosis. Recent study demonstrates that sustained physiological Hyperinsulinemia commonly seen in fasting state in Type2 Diabetes Mellitus or obesity, significantly alters the intracellular partitioning of glucose between oxidative & non-oxidative pathways. In muscle, glucose disposal is markedly reduced. This impairment of non-oxidative glucose disposal contributes to insulin resistance. Recently it has been suggested by Michael stern et al that Type₂ Diabetes Mellitus and CHD share common genetic and environmental factors. The consequence of inflammatory disease in response to oxidative insult is the Common soil for both the diseases.

Oxidative stress also forms a Common soil for Type₂ Diabetes Mellitus / CHD forming oxygen free radicals which are toxic metabolites derived from refined cereals. Hyperglycemia and insulin resistance and hyperinsulinemia may be associated with increased production of oxygen free radicals glucose autooxidation and increased non-enzymatic glycation. Oxidative stress is also responsible for increased platelet reactivity and thromobogenesis. This newer concept of "Common soil" for both Type₂ Diabetes Mellitus and CHD promise a simple therapeutic option for dietary intervention.

ii) IMPACT OF DYSLIPIDEMIA²⁵

The nexus between dyslipidemia and CAD in diabetic patients is overwhelming. An insight into this nexus is mandatory to understand this deadly complication.

Current knowledge suggests the possible role of hypertriglyceridemia, low levels of high density lipoprotein cholesterol as important risk factors. At present atherosclerotic vascular disease is the most common complication of Diabetes Mellitus, particularly in women. Incidence of CAD has been found to be 2 to 3 fold higher amongst patient with Diabetes Mellitus. Analysis by Das. S, Mishra R.K, Jena et al, over the past three decades indicates progressive increase in the incidence of CAD as the cause of deaths.

The genesis and progression of atherosclerotic lesion have been observed to be different in diabetic patients. Normally it is 'Type 1' or Rokintansky Type' lesion. But in Diabetes Mellitus it is "Type 2' or 'Virchow's Type' where the susceptibility for atherosclerotic vascular disease is much greater.

Glycation of apoproteins, cell membrane proteins and proteins of membrana propria of the endothelium along with raised quantity of glucosaminoglycans and advanced glycation end products in Diabetes Mellitus enhances LDL, VLDL. They bind the endothelial cells and accelerates the atherosclerosis. Further the enzyme Lecithin - Cholesterol Acyl Transfarase (L-CAT) regulating conversion of free cholesterol to esterified - cholesterol in circulation as well as lipoprotein lipase (LPL) present at capillary endothelium. Insulin primes these enzymes and thus insulin resistance and insensitivity existing in the peripheral bed of Type₂ Diabetes Mellitus can dampen these process. Suppression of L-CAT activity will lead to lesser generation of HDL while slower action of LPL will produce delayed clearance of VLDL.

In diabetes due to loss of endothelial electrical barriers, consequent to glycation of basement membrane protein, TG rich LPs get access to sub-endothelial layers and cause lipid ladening of smooth muscle cells - the key to atherogenesis. Diabetic subjects seen in India have high level of Triglycerides (TG). Raised TG with decreased HDL-c and normal or near normal TC and LDL is the usual lipid profile in diabetic patients. Raised TG even in the presence of normal TC and LDL-c is atherogenic.²⁶

(iii) IMPACT OF DIET AND ANTIOXIDANTS²⁷

Diabetes is commonly associated with CAD because dyslipidemia, oxidative stress and hyper insulinemia are related to diet and life style. Obesity in Type₂ Diabetes Mellitus increases the risk of CAD. Diet high in plant foods and low in fat are associated with a lower prevalence of Diabetes and CAD. Besides total and saturated fats and refined carbohydrates which adversely affects diabetes, the n6/n3 ratio of fatty acids and antioxidants are being increasingly linked with atherosclerosis.

Antioxidants Vitamins A, E and C, beta - carotene, dietary soluble fibre, starch foods, plant sterol, flavanoids polyphenols, and ubiquinone may have beneficial effect by decreasing oxidative stress²⁸. Low fat cereal based diets are associated with low risk of Diabetes and CAD in rural population of India. Table 1 shows sources of free radicals in DM.

SOURCES OF FREE RADICALS IN DIABETES

S.No	Particulars
1.	Non-enzymatic glycosylation of proteins
2.	Changes in the level of inflammatory mediators
3.	Monosaccharide auto – oxidation.
4.	Polyol pathway activity and alterations in sorbital pathyway activity
5.	Indirect production of free radicals through cell damage by other causes
6.	Reduced antioxidants reserve
7.	Metabolic stress due to change in energy metabolism.

Table - 1

Free radical scavengers which limit hydroxyl radical by trapping reactive radicals in both hydrophilic and lipophilic cell membrane environments are vitamin C gluathione and uric acid and vitamin E and ubiquinol respectively. ubiquinol is a direct free radical scavenger as well as it also regenerates vitamin E after it converts into inactive tocopheroxyl form. Vitamin E is a highly potential antioxidant.

ANTIOXIDANT DEFENCES IN THE BODY

Particulars	
Primary enzymatic defences	Functional dependences
Super oxide dismutase	Zinc, Manganese
Glutathione peroxidase	Selenium
Catalase	Iron Copper
Coenzyme Q (Ubiquinol)	
Secondary stabilizing substances	
Vitamin E	Miscellaneous
Vitamin C	Albumin
Vitamin A	Uric acid
Beta – Carotene	Transferrin
Carnitine	Ceruloplasmin

In brief, eating 400 gm / day of fruits, vegetables and legumes and another 400 mg/day of cereals in conjunction with 25 gm/day of oils, increased physical activity (>300 K cal/d) and cessation of smoking may be protective in the primary prevention of diabetes and CAD. There is great evidence that as pancreatic betacells and arterial endo-thelial cells have low antioxidative enzyme activities, they might be more sensitive to free radical injury.

EVALUATION OF MYOCARDIAL ISCHEMIA

Coronary artery disease (CAD) is common in diabetic patients, Besides epicardial coronary arteries, microcirculation is also involved. CAD is a common cause of premature morbidity and mortality in diabetics Evaluation of myocardial ischemia in them is always a key issue. Early detection is therefore paramount importance. In a Patient with known CAD, of the total ischemic episodes only 20-30% are symptomatic and the remaining 60-70% are silent known as Silent Myocardial Ischemia (SMI). Clinically CAD is evaluated by several tests besides meticulous history and physical examination.

S.No	Particulars
1.	Resting ECG
2.	Ambulatory ECG monitoring
3.	Stress test
	a) Stress Electrocardiography ²⁹
	b) Stress Echo
	c) Pharmacological Stress Echo
	d) Stress Thallium
4.	Coronary angiography ³⁰
5.	Intracoronary vascular ultrasound (selected cases)

1. Resting ECG

In diabetic patients resting ECG is normal in approximately half of the patients. The most common ECG abnormalities are non-specific ST-T change with or without evidence of prior MI. A normal ECG doesn't exclude the presence of CAD. Severe (Triple Vessel disease) 3° VD may exist with a normal resting ECG.

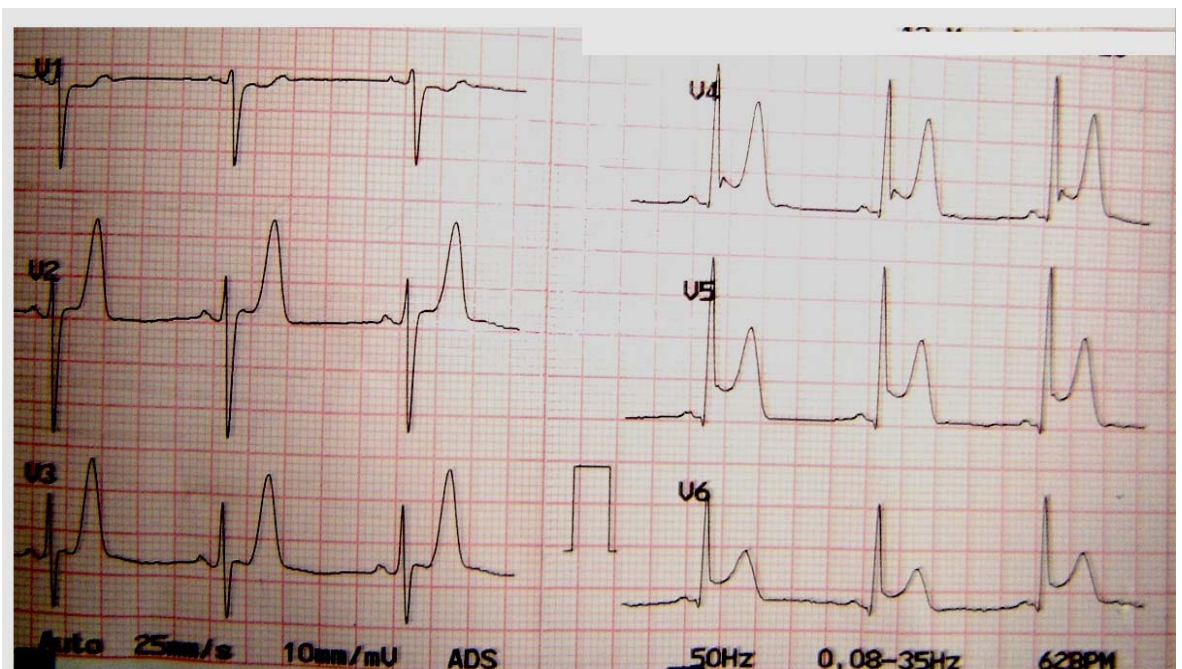
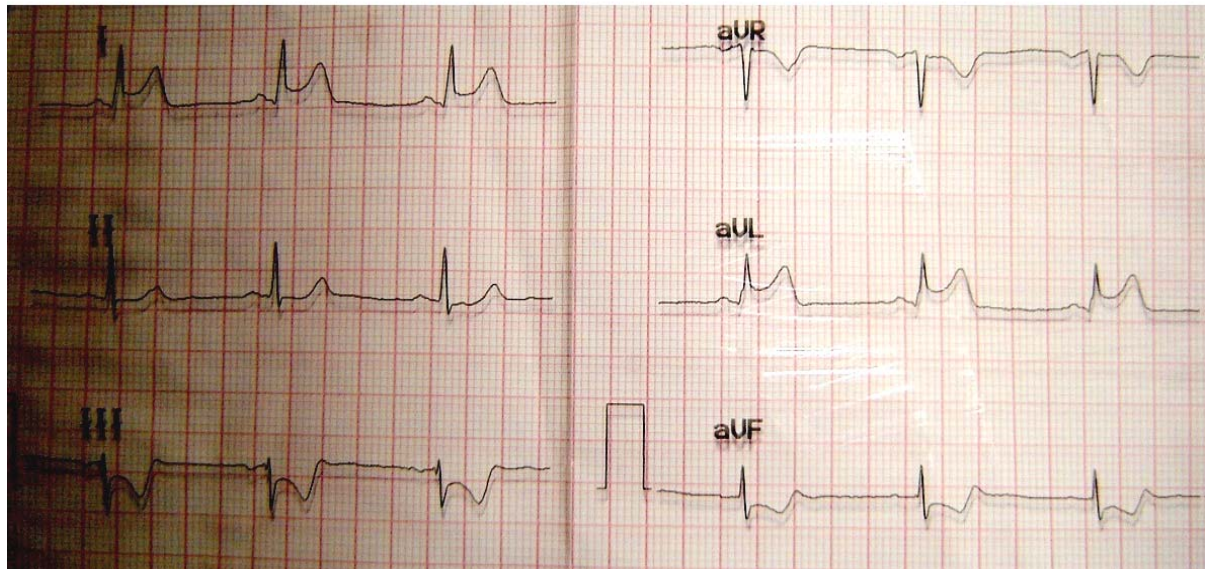
A variety of conduction disturbances like left anterior fascicular block or LBBB may be seen in asymptomatic diabetic patients. LVH on ECG may suggest associated hypertension. Q wave or non Q MI may be seen in ECG as a silent findings.

2. Ambulatory 24 hrs Electrocardiography (HOLTER'S)

It is used to detect SMI or ventricular arrhythmias in diabetic patients with suspected or established CAD and myocardial ischemia. Cardiac autonomic neuropathy may mask anginal episodes SMI and total Ischemic burden.

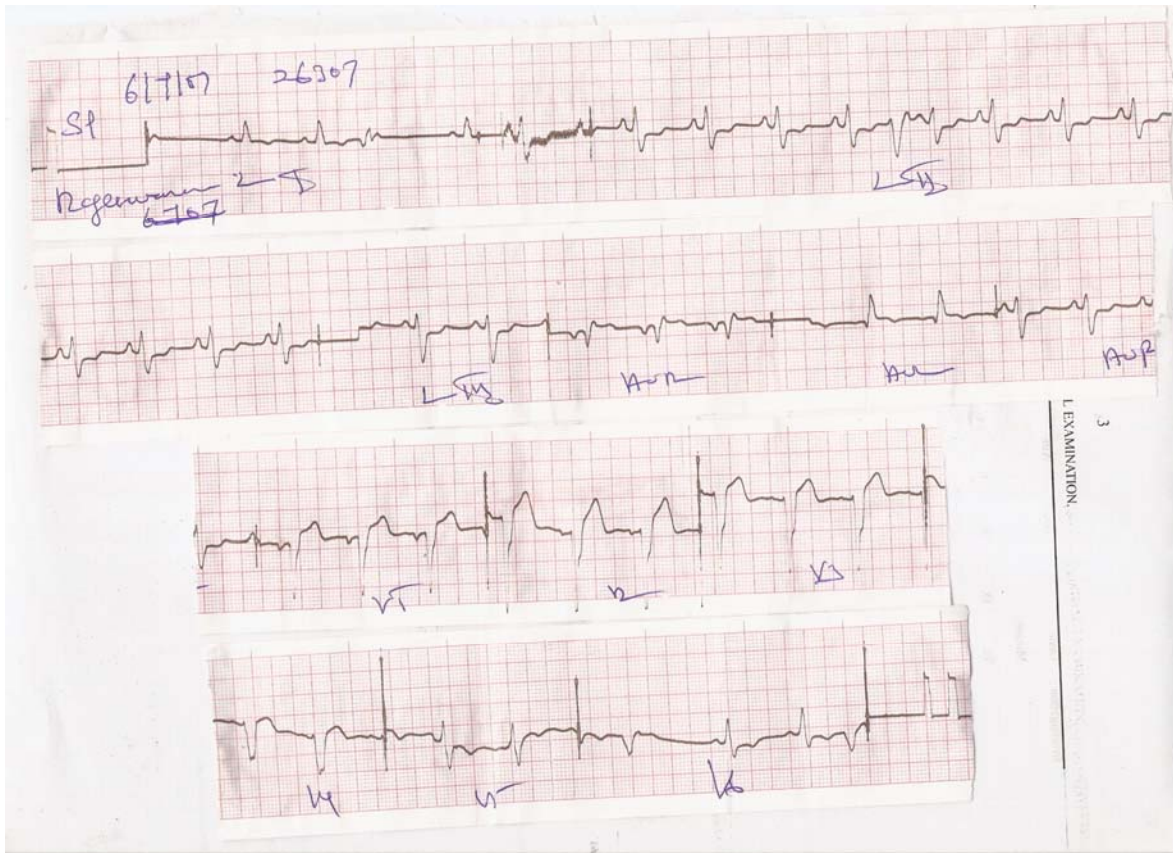
ECG SHOWING ANTEROLATERAL MYOCARDIAL INFARCTION

Fig1



LBBB WITH MI

Fig 2



3. Stress Test

a) Stress Electro Cardiography:

Tread Mill stress test or bicycle ergometric stress test is used as a screening test for CAD in symptomatic or asymptomatic diabetes. It is safe, simple and inexpensive. Tread mill exercise testing may suggest the presence of significant obstruction in one or more coronary arteries. It is 66% negative in 1° VD (1 Vessel Disease). 33% negative in 2° VD. All 3° VD are virtually picked up by this test.

The Tread mill test is useful particularly in 2-vessels and 3-vessels disease during exercise and recovery. The predictive value is high if typical chest pain occurs during the exercise with ST segmental depression - horizontal or downsloping of 1 or more than 1 mm. The early onset of ST segment depression during exercise testing in diabetes, its long persistence following discontinuation of exercise, a down sloping or horizontal depression and a low work capacity or duration of exercise are all strong indicators of multi vessel CAD with myocardial ischemia in diabetes. A negative result does not exclude CAD. However, the presence of three vessel or left main CAD is excluded in diabetic patients by negative test.

b) STRESS ECHO CARDIOGRAPHY

2 Dimension Echocardiography and Doppler echocardiography is very valuable, relatively inexpensive and has high sensitivity and specificity when performed immediately or during stress to detect LV regional wall motion abnormality and LV function.

c) PHARMOCOLOGICAL STRESS ECHOCARDIOGRAPHY:

Among diabetic patients unable to exercise, stress echo is done with Dobutamine or Dipyridamole. Dobutamine is preferred because of its biphasic response. Dobutamine is used in initial dose of 5 $\mu\text{g}/\text{Kg}/\text{min}$. The dose is increased by 5 $\mu\text{g}/\text{Kg}$ every 3rd minute till a maximum dose of 40 $\mu\text{g}/\text{Kg}/\text{min}$ is used or target heart rate is achieved or an adverse response is seen.

d) STRESS THALLIUM IMAGING

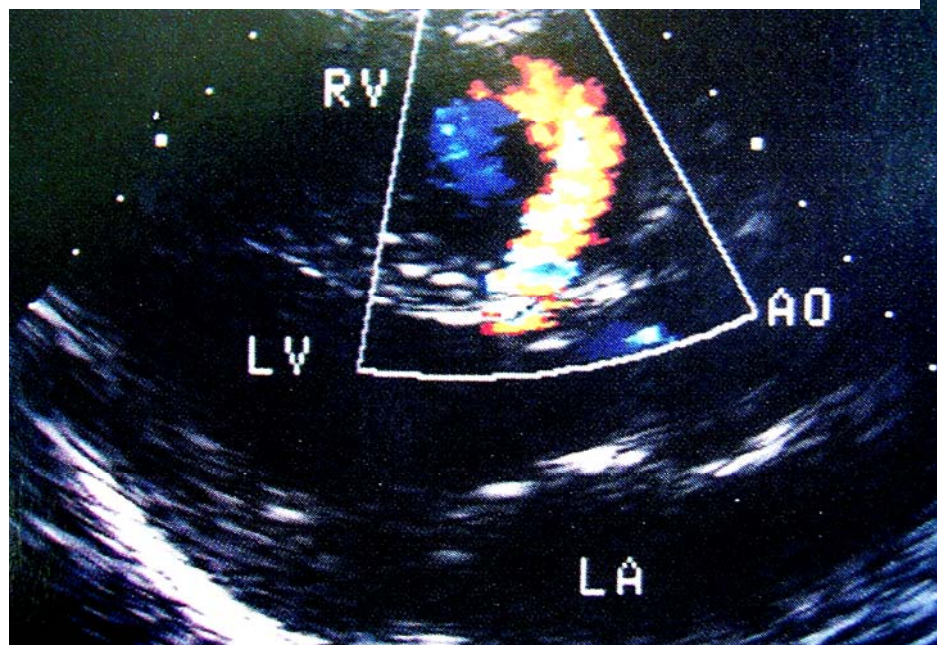
In individuals with negative stress ECG and high suspicion of CAD, stress thallium imaging is performed. The test has sensitivity of 82% and specificity of 88%.

45 years old male patient with acute myocardial infarction and pulmonary edema

Fig 3



ECHO SHOWING MITRAL REGURGITATION AFTER MI
Fig 4



4.CINE CORONARY ANGIOGRAPHY

i) Coronary Angiography³¹

Definitive diagnosis of CAD, its precise assessment and anatomic severity requires coronary angiography. This test is very valuable, definitive and essential when stress test is positive and or left ventricular function is impaired clinically or non invasively. This helps in planning suitable revascularization in patients with ischemia. The diameter of stenosed vessel in angiography in DM patients have a significantly smaller luminal diameter in Infarct Related Artery than in non - DM patients.

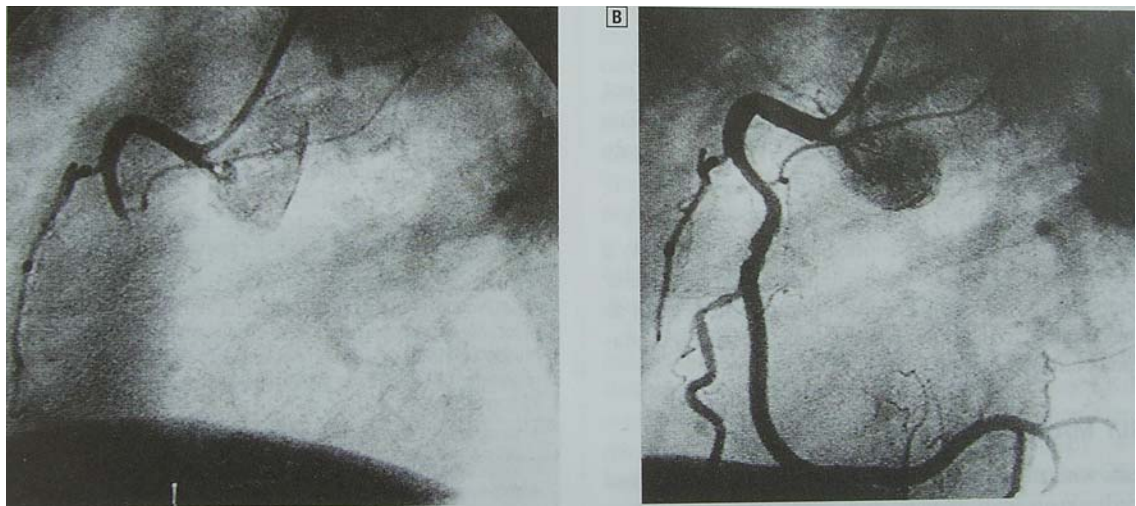
The angiographic restenosis rate following PTCA reported in National Heart, Lung, Blood Institute Angioplasty Registry is 49% to 71% in diabetics. Newer devices like DCA stenting and excimer laser angioplasty have failed to reduce restenosis³². Re-occlusion rates following lytic therapy also higher than in non-diabetics³³.

ii) Intracoronary Vascular Ultrasound (IVUS)³⁴

In selected cases of CAD where there is controversy regarding severity of atherosclerotic obstruction on coronary angiography, the coronary artery patho-anatomy is evaluated with Intracoronary Vascular Ultrasound.

**CORONARY ANGIOGRAM AND SCHEMATIC DIAGRAM SHOWING BLOCK IN LEFT
ANTERIOR DESCENDING ARTERY**

Fig 5



Coronary angiography no doubt remains the modality of choice for rapid visualisation of the entire coronary tree. However, it should be realised that it only exhibits luminal narrowing as a two-dimensional silhouette of a three-dimensional structure, Intracoronary Vascular Ultrasound is utilised to unmask atheromatous disease not properly visualised or missed by coronary angiography. IVUS although is not widely available, it is the modality of choice to judge the adequacy of coronary interventions.

IVUS is capable of providing high resolution cross-sectional images of normal and abnormal arterial architecture, atheromatous plaques and its complications and is radically changing the diagnosis and interventional management of coronary artery disease³⁵.

Advantages of IVUS

S.No	Particulars
1.	Superb display of circumferential extent of atheroma.
2.	Unmasks atheromatous disease not visualized by coronary angiography
3.	Visualization of plaque, its distribution and morphology,
4.	Demonstrates presence of dissection, intimal flaps
5.	Judges adequacy of coronary interventions in a better way.

ACUTE MYOCARDIAL INFARCTION IN DIABETES

MELLITUS³⁵

The clinical presentation and course of AMI in DM are different.

³⁶AMI occurs earlier in diabetic patients, and has higher incidence of silent infarct and pump failure. Relative risk of myocardial Infarction is 50%³⁷ greater in diabetic men, 15% greater in diabetic women.

³⁸Sudden death 50% more often in diabetic men. 300% more often in diabetic women. 30% of death in diabetics are as a result of AMI. Increased platelet aggregation due to synthesis of thromboxane A₂ in high amounts in poorly controlled diabetes mellitus and vascular spasm which leads on to thrombus formation in vessels with its sequelae. Since platelet aggregability is high through out the day – MI³⁹ occur at any time.

SILENT MYOCARDIAL INFARCTION:⁴⁰

Ischemic chest pain is blunted in Diabetes Mellitus. Myocardial ischemia or infarction may be associated with mild symptoms or totally absent. During Tread Mill exercise test, angina is absent due to severe autonomic neuropathy. Atypical symptoms like dyspnoea, fatigue, nausea, vomiting or confusion which are attributed to metabolic disturbances leading to delay in the diagnosis. Periinfarction mortality is high.

COMPLICATIONS OF MYOCARDIAL INFARCTION IN

DIABETES MELLITUS

Diabetic patients develop complications more often and mortality may be high. These include cardiogenic shock, CHF, conduction disturbances, etc. Anterior wall infarction is more common. On occasion Left⁴¹ Ventricular Free wall rupture may be the presenting feature. Reinfarction is higher. Early introduction of Aspirin, thrombolytics and Beta Blockers reduces mortality^{42,43}. Coronary artery Disease is the most common cause of death in Diabetes Mellitus Patients.^{44,45}

HYPERTENSION IN DIABETES MELLITUS

EPIDEMIOLOGY

Diabetes and hypertension Co-exist three times more commonly, strongly predispose the individual to atherosclerotic cardiovascular disease. Essential hypertension with Type₂ Diabetes Mellitus constitute over 90%. Diabetic nephropathy which occurs after 15 years of diagnosis of diabetes is one third of those with Type₁ Diabetes Mellitus and 20% of those with Type₂ Diabetes Mellitus. Isolated systolic hypertension is observed more commonly in diabetics and supine hypertension with orthostatic hypotension observed in diabetics with autonomic neuropathy⁴⁷. Type₁ Diabetes Mellitus patients are usually normotensive until overt renal disease develops.

ETIOLOGY

Hypertension in diabetic patients can be classified into four different heads.

a. Renal hypertension with diabetic renal disease.

Type₁ Diabetes Mellitus patients are usually normotensive until overt renal disease develops. In Type₂ Diabetes Mellitus, hypertension increases markedly even before the proteinuric state. Micro albuminuria is the first sign of diabetic renal disease.

b. Hypertension without renal disease

Factors that contribute to the development of this type of hypertension are genetic, obesity, glucose intolerance, hyperinsulinemia etc. Insulin causes reabsorption of sodium from distal part of nephron and has antidiuretic effect.

c. Systolic Hypertension

Isolated systolic hypertension may be defined as a systolic BP of 140 mm of Hg or more with normal and low diastolic pressure. In diabetic patients with atherosclerosis, large arteries lose their elasticity and gradually become rigid and all blood ejected from the left ventricle will enter this arterial system which is incapable of expansion, thus systolic pressure will be increased.

d. Orthostatic Hypotension

Baroreceptor reflexes which are important in maintaining blood pressure are often blunted in patients with diabetic neuropathy. Other mechanisms include loss of proteins in urine causing contraction of plasma volume and decreased myocardial contractility and decreased cardiac output.

PATHOGENESIS

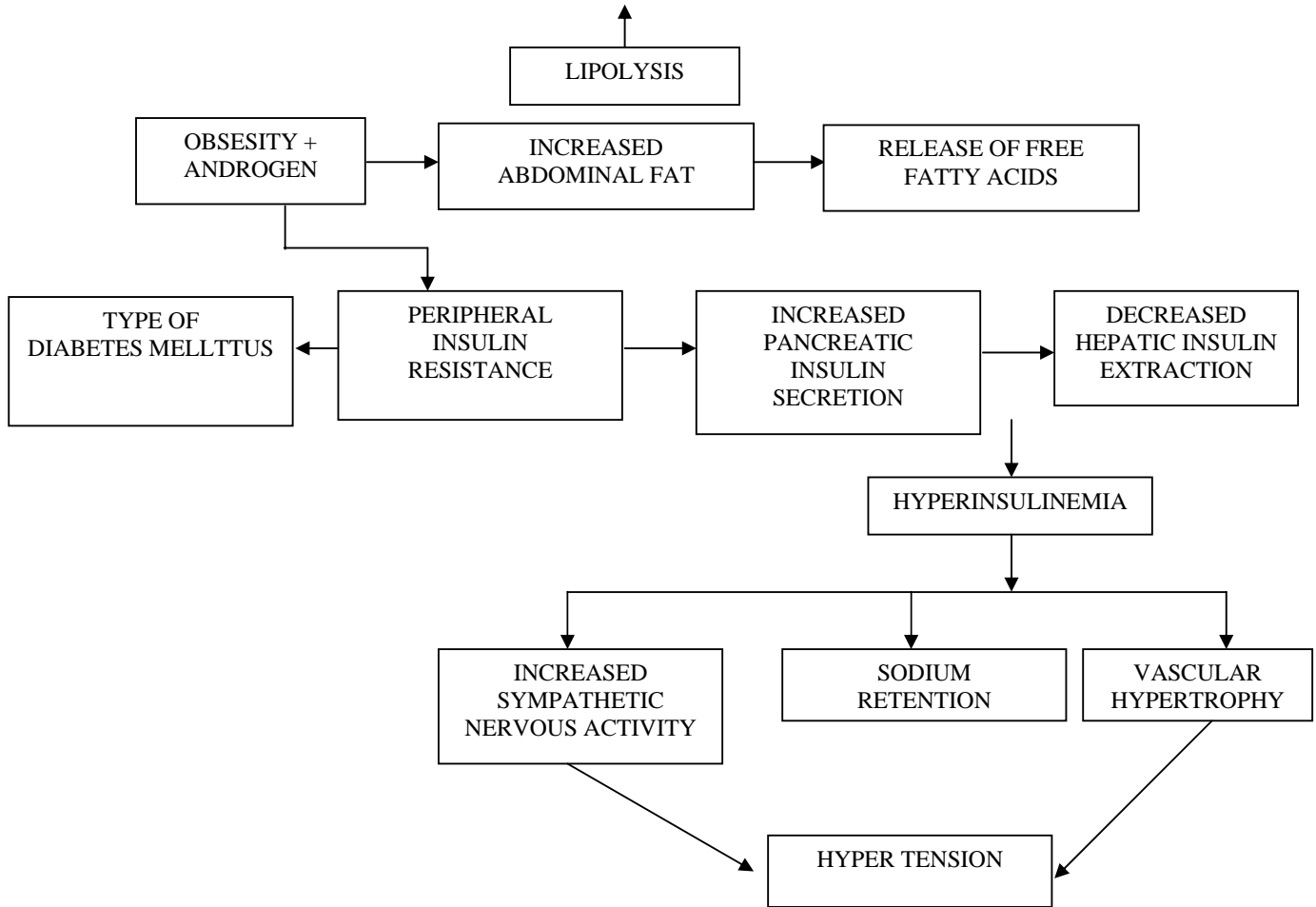
The genesis of hypertension in diabetes mellitus include renal disease, hyperinsulinism, atherosclerosis, genetic etc. The role of genetic predisposition is suggested by the fact that history of hypertension is four times more frequent in patients of Type₁ Diabetes Mellitus with nephropathy than those without nephropathy. An increase of Sodium / Lithium counter transport by the red blood cells is considered a marker for essential hypertension⁴⁹. Proposed mechanisms by which insulin resistance and or hyper insulinemia may lead to hypertension in obese patients are.⁵⁰

1. Enhanced renal sodium and water reabsorption.
2. Increased salt intake and salt sensitivity.
3. Augmentation of the pressor and aldosterone response to angiotensin II.
4. Changes in Trans membrane electrolyte transport, increased intracellular sodium and Ca accumulation. Decreased Na^+ , K^+ - ATP ease activity.
5. Stimulation of sympathetic nervous activity.
6. Reduced synthesis of vasodilator prostaglandins.
7. Impaired vasodilatation.
8. Increased secretion of endothelin.

The relation between hypertension, diabetes and obesity predominantly in the upper body, composing the major components of the insulin resistance 'Syndrome X' is shown in the following algorithm.

SYNDROME X⁵¹

HYPERTRIGLYCERIDEMIA



Atherosclerosis is premature, more extensive and has accelerated progression associated with abnormalities of lipid metabolism because of lack of insulin which lead on to faulty LDL, receptor and accumulation of LDL. This LDL gets transported subendothelially resulting in lipid per oxidation and circulating T- lymphocytes and monocytes adhere to vascular endothelium. These cells take up lipids and lipoproteins thus becoming foam cells forming fatty streaks projecting into the lumen of blood vessels. Hyperglycaemia increases endothelin secretion which is a powerful vasoconstrictor and mitogenic factor, while production of Nitrous oxide is diminished (a vascular smooth muscle relaxant, antimitogenic which inhibits platelet adhesiveness). Thus smoking, insulin resistance, hyperlipidemia and genetic predisposition, accelerates atherogenesis and hypertension occurs early in diabetes.

MEDICAL THERAPY

1. DIET⁵²

Dietary fats and antioxidants have significant role in modulating insulin action and secretion. Faulty nutrition of the present day urban society present epidemic Type₂ Diabetes Mellitus and CAD. Preservatives and pesticides in processed foods add there own quota of oxygen free radicals (OFR).

Unsaturated fatty acids of both n6 and n3 in their physiological disproportion with high n6 / n3 ratio promote a variety of immune inflammatory disorders including Diabetes Mellitus and atherosclerosis.

Guidelines for diet: ⁵³

1. Slow weight reduction of about 2 Kg /month can be achieved with a reduced calorie intake of about 20 Kcal/Kg.
2. Importance of fish in the diet to have 5 or more helpings (each 30 gms) a week.
3. To avoid increased availability of arachidonic acid avoid other animal foods.
4. Fresh fruits 2 – 3 helpings of vegetables at each meal. Almost all vegetables and pulses can be taken. Avoid simple sugars, refined cereals and bakery products.
5. Alcohol to be avoided.
6. Saccharin can be used in small quantity.

2. ALCOHOL^{53a}

Alcohol in small amounts (less than two units, a unit is 9 Gin- equal to 1/2 pint of beer or 8 Gm of wine or 30 ml of spirit) a day called Sensible Drinking is believed to provide protection from coronary mortality and atherosclerosis, but in large amounts increases blood pressure and excess cardiac events. This is known as 'U' 'J' or 'J'; shaped response.

3. DYSLIPIDEMIA

When diabetes and dyslipidemia occurs in combination, the risk is significantly increased in Type₂ Diabetes Mellitus patients, whereas insulin therapy corrects lipid abnormalities in Type₂ Diabetes Mellitus patients.

Management of dyslipidemia in Type₂ DIABETES MELLITUS with CAD is by step care approach.

Step 1	<p>Non pharmacological measure</p> <p>Diet therapy</p> <ul style="list-style-type: none"> • energy restriction • composition • cessation of smoking, reduction of alcohol intake, increased physical activity • aerobic exercise, brisk walking, swimming. bicycling, yoga etc
Step II	<p>Hypoglycemic drugs</p> <ul style="list-style-type: none"> • Sulfonylurea • Biguanides • Insulin • Antioxidants – Vitamin C, - Carotene, Vitamin E.
Step III	<p>Lipid lowering drugs</p> <ul style="list-style-type: none"> • Statins • Bile acid sequestrants • Nicotinic acid • Probucol • Estrogens

Although conventionally non - pharmacological treatment in conjunction with good glycemic control is usually tried for 3-6 months for managing dyslipidemia in diabetes, in CAD with malignant progression, drug therapy should be started concurrently.

**CURRENT RECOMMENDATIONS FOR DESIRED
LEVEL OF CHOLESTEROL:⁵⁵**

	Primary Prevention	Secondary Prevention
Lipid Determination	Desirable	Desirable
Total Cholesterol	< 200 mg/dl	
LDL Cholesterol	<130 mg/ dl	<100gm/dl
Among Indian	<100 mg/dl	<80 mg/dl
HDL Cholesterol	>40 mg/dl	>40 mg/dl
TC/HDL –C Ratio	<4.5	<3.5
LDL-C/HDL-C Ratio	<3	<2.5
Triglyceride	<150 mg/dl	<150 mg/dl ⁵⁶

Hypertension is frequently associated with diabetes. The drugs which have an adverse effect on lipids should be avoided. ACE inhibitors, calcium channel blockers and alpha-1 antagoins can be used as they are lipid friendly and do not cause carbohydrate intolerance.

4.Anti Diabetic Drugs

The bulk of CHD in diabetic patients belongs to Type₂ Diabetes Mellitus and it is in this subject, the choice of drug may vary from one drug to another or to a combination of drugs. In MRDM and FCPD patients the choice to antidiabetic drug is limited to insulin only. In Type₂ Diabetes Mellitus the choice of drugs may vary. The specific steps in the management of CHD are practically similar for subjects with or without diabetes. The second generation agent gliclazide has been described to have special properties for prevention of atherosclerosis and the sequelae by some other mechanisms in addition to control of hyper glycemia. So it is considered more suitable for use in elderly with or without CHD.

The novel Sulfonylurea agent Glimipride⁵⁷ – its action on cardiac myocytes and coronary vasculature is very much lower than others and acts by combining with a different protein at a portion of the receptor different from other SU compounds. It has antiatherogenic and anti-thrombotic properties. Biguanides are specially suited for obese patients with CHD without hepatic or renal dysfunction or cardiorespiratory insufficiency. The antiatherogenic effect and favourable action on haemorrheology further adds to their utility in CHD. Metformin is preferred.

Insulin is the preferred agent in Type₂diabetes Mellitus with AMI and also in post MI state. Hyperglycemia is controlled by low dose insulin infusion with regular .blood glucose monitoring so as to maintain the levels between 100-150 mg/dl. So diabetes mellitus should be under treated as hypoglycemia is worser than hyperglycemia⁵⁸.Alpha glucosidase inhibitors and soluble fibres are suitable for use particularly in overweight patients with high post-prandial plasma glucose. Thiazolidinediones lower insulin resistance and hence may be suitable for use in diabetic patients with CHD.

Control should be achieved with low to moderate doses or else combination have to be taken. Insulin should be started when fasting plasma glucose is above 15 mmol/L and in very lean patients. Any anti hypertensive can be used. But Beta blockers are usually avoided because it decreases the release of insulin and masks the symptoms and signs of hypoglycaemia. ACE inhibitors are more preferred. It can prevent development of nephropathy and control hypertension.

INTERVENTIONAL TREATMENT

1. Catheter interventions⁵⁹

Catheter interventions like angioplasty, intracoronary stents and others are being done in diabetes mellitus patients with CAD. Restenosis is higher in insulin requiring patients and they have reduced long term survival. Both PTCA and CABG have same long terms survival with poor outcome after bypass surgery.

2. Surgical revascularization^{60,61}

"With technological advances and refinements in techniques of surgery, the presence of a diabetic state does not come in the way for providing benefit of surgical revascularization. Present day revascularization procedures include.

(i) Conventional CABG through mid sternotomy approach^{62.}

This may be performed by using

- a) Saphenous vein graft
- b) Internal mammary artery
- c) Total arterial revascularization using IMA, Radial, Gastroepiploic or inferior epigastric artery.

(ii) Transmyocardial Lazer revascularization (TMLR).

In this procedure vascular channels are created from left ventricular cavity directly into the myocardium (based on anatomy of reptilian heart). Frequent indication for this procedure is refractory angina where PTCA or by pass surgery is not allowed. TMLR can be combined with CABG in patients in whom by-pass surgery cannot be performed for all area because of prevalence of diffuse coronary artery disease is higher on diabetics. This procedure is well tolerated in diabetic patients and showed no significant adverse outcome compared to non-diabetics.

(iii) Minimally invasive direct coronary artery bypass (MIDCAB)

This technique performed with small invasion and without cardiopulmonary bypass, and has dramatically changed the surgical management of coronary artery disease.

RISK FACTORS IN DIABETES MELLITUS

Obesity:

Obesity is defined as Body Mass Index of ≥ 30 . All the patients were classified according to their BMI

Classification	BMI
Underweight	<18.50
Normal	18.50 – 24.99
Overweight	> 25.00
Pre-obese	25.00-29.99
Obese Class – I	30.00 – 34.99
Obese Class – II	35.00 – 39.99
Obese Class – III	> 40.00

Family History:

Family History is recorded as a risk if there is a history of coronary artery disease in Male first – degree relative of <55yrs or in female first-degree relative if <65 yrs.

Dyslipidemia:

Dyslipidemia is defined as LDL cholesterol level of > 100 mg/dl (or) HDL cholesterol < 40 mg/dl (or) non-HDL Cholesterol > 130 mg/dl. Non-HDL Cholesterol = Total Cholesterol – HDL Cholesterol.

Smoking History:

Patients were recognized as a non-smoker if they never smoked or they gave up smoking > 15 years back, an ex-smoker if they stopped smoking for > 3 months, and as a Current smoker if they still smoked within last 3 months. (After 15 years of cessation, the risk of a new Myocardial Infarction in former smokers is similar to that for those who have never smoked)

Materials and Methods

MATERIALS AND METHODS

SELECTION OF PATIENTS

100 diabetes mellitus patients who attended CMCH for Hypertension and Ischemic heart disease in ICU and medical wards were included in this study over a period of one year. All the patients were subjected to detailed careful clinical examination and laboratory investigations to assess cardio vascular status.

METHODS OF EXAMINATION

Detailed clinical examination including JVP, pulse, position and characters of cardiac apical impulse was done with an enquiry about the risk factors. BP was recorded in supine, standing and sitting positions in both upper and lower limbs. The heart was auscultated for abnormal S3, S4 and gallop rhythm. Fundus examination was done for changes of hypertension & diabetes. The respiratory system was examined for any adventitious sounds. Abdomen was examined for organomegaly, evidence of free fluid and bruit. Central nervous system was examined for any neurological deficit.

Body mass index was calculated from the formula (weight in kilogram / height in meter'.)

$$\text{BMI} = \frac{\text{Weight in Kg}}{\text{Ht in m}^2}$$

The following investigations were done.

1. Urine for albumin, sugar, deposits & acetone.
2. Blood sugar & urea.
3. Serum - Creatinine, cholesterol and electrolytes.
4. Lipid profile - Total cholesterol, LDL, HDL, VLDL,, TG.
5. X – Ray chest PA view
6. ECG
7. Echo cardiogram

CRITERIA FOR DIAGNOSIS

1. Diabetes mellitus

The criteria proposed by the American Diabetes Association was followed for the study.

a. Unequivocal elevation of plasma glucose concentration together with the classical symptoms of diabetes such as polyphagia, polydipsia and weight loss.

b. Criteria for Diagnosis of DM:

1. Symptoms of DM plus Random Blood Sugar Concentration \geq 200mg/dl. (or)
2. Fasting Plasma Glucose $>$ 126mg/dl (or)
3. 2hrs plasma glucose \geq 200mg/dl during an oral glucose tolerance test.

2. Ischemic Heart Disease:

a. History :

Typical - with pain or discomfort localised in the upper or mid sternal region and appearing on effort. Pain may radiate to the jaws, shoulders, or arms on one or both sides with crushing or constricting in character compels the patient to halt all activity. Atypical - asymptomatic or is characterised by symptoms such as severe breathlessness, profound weakness, sudden loss of consciousness or acute pulmonary oedema.

b. ECG Changes

- i. Ischaemia :* ST segment depression of 1 mm from the J point with or without inversion of T wave.
- ii. Infarction:* Q-wave infarction - presence of pathological Q wave depth is more than 25% of the corresponding R wave and width is more than 0.04 sec, with ST segment elevation and T wave inversion.

Non. Q wave infarction:

Anatomic Zone	ECG Zone
1. Inferior wall	II, III & a VF
2. Anteroseptal wall	VI to V4
3. Extensive anterior wall	I, aVL, VI to V6
4. Apicolateral	I, a VL, V4 to V6

Hypertension

If the blood pressure is more than 140/90 mm of Hg for three consecutive visits is diagnostic of systemic hypertension.

Data Analysis of Results

ANALYSIS OF RESULTS

The data collected out of the 100 patients there were 73 males and 27 females.

The Observations are as follows:

1. Number of patients with Ischaemic heart disease, including myocardial infarction.

Total no of IHD patients = 57

Ischaemic patients -31 (54.3%), Infarction patients - 26 (45.6%)

Both ischemia and infarctions -7.

Silent ischemia -9 (29.03%), Silent infarction - 11 (42.3%)

2. Number of patients with hypertension without

associated coronary ischemic heart disease = 21

3. DM patients without IHD / SHT = 22

4. Number of patients with both systemic hypertension

and ischemic heart disease in Diabetes Mellitus = 33

5. The largest number of Ischaemic Heart Disease 6-10 years

after the onset of DM is = 23

6. The 'risk factors' such as smoking, alcoholism,

hypertriglyceridemia and family history contribute

maximum for development of IHD and Systemic

Hypertension in Diabetes Mellitus i.e = 62 %

VARIOUS TYPES OF CARDIOVASCULAR
COMPLICATIONS IN
DIABETES MELLITUS

S.No	Total No. of Diabetic patients studied	Type of Cardiovascular disease	No. of patients affected by the disease	Percentage
1.	100	No IHD	22	22
2.	100	SHT Only	21	21
3.	100	IHD Only	24	24
4.	100	SHT + IHD	33	33
5.	100	Total IHD	57	57
6.	100	Total SHT	54	54

As shown in the table No. 1

- ✓ In the present study, 57% have IHD.
- ✓ 54 % have systemic hypertension.
- ✓ 33 % have both hypertension and ischemic heart disease.

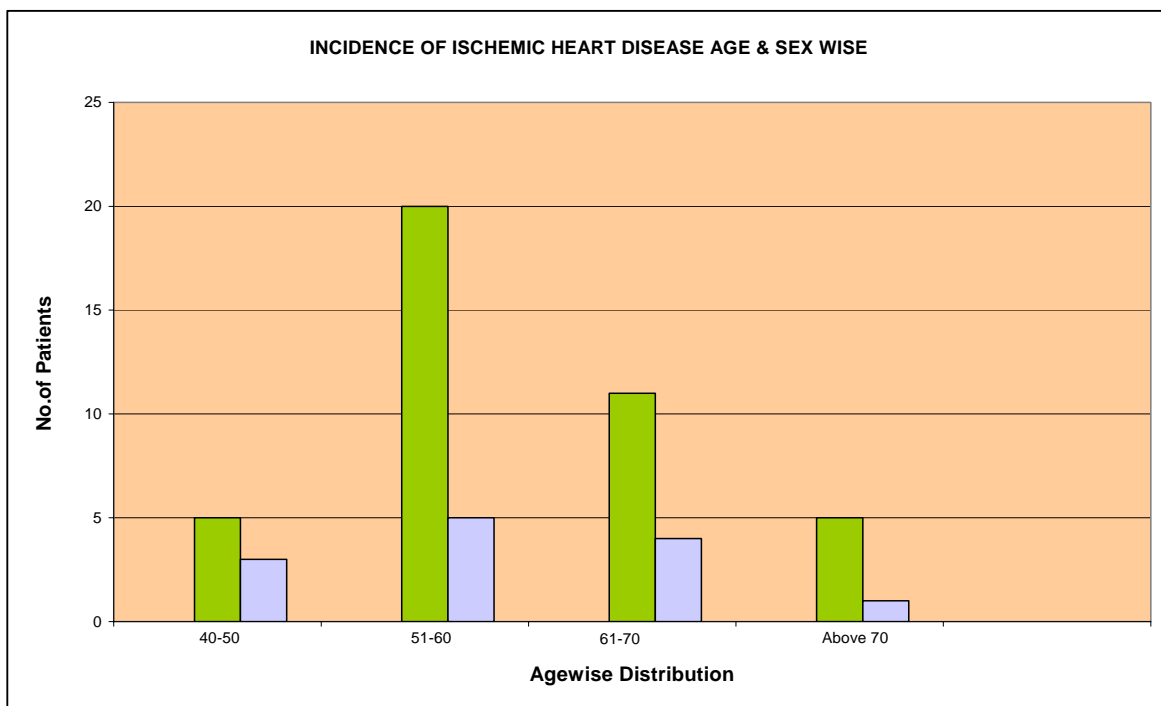
INCIDENCE OF ISCHEMIC HEART DISEASE

AGE & SEX WISE

S.No	Age in years	Sex		Total	Percentage
		Male	Female		
1	40-50	6	2	8	14.03
2	51-60	16	3	19	33.33
3	61-70	15	3	18	31.57
4	Above	11	1	12	21.05
		48	9	57	

INCIDENCE OF ISCHEMIA AND INFARCTION

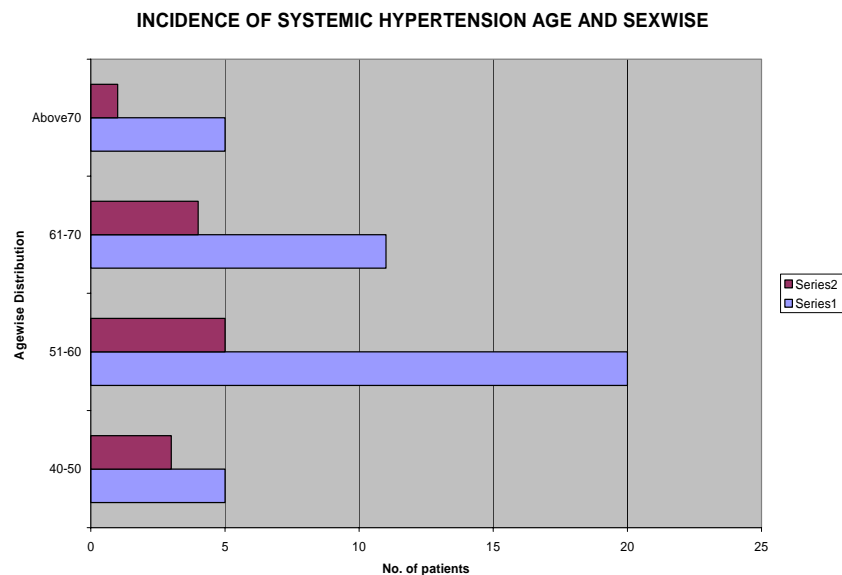
S.No	Type of Lesion	No.of Patients	Percentage
1	Ischemia	31	54.3
2	Infarction	26	45.6
3	Both Ischemia and Infarction	7	12.28



INCIDENCE OF SYSTEMIC HYPERTENSION **AGE AND SEXWISE**

Age in years	Sex		Total	Percentage
	MALE	FEMALE		
40-50	5	3	8	14.81
51-60	20	5	25	46.29
61-70	11	4	15	27.77
Above 70	5	1	6	11.11
	41	13	54	

The largest number of systemic HT were in the age group of 51 – 60 yrs, i.e., 25 cases (46.29%). The maximum sufferers are males 41



INCIDENCE OF ISCHEMIC HEART DISEASE

AND SYSTEMIC HYPERTENSION

Age in years	Sex		Total	Percentage
	Male	female		
40-50	4	2	6	18.18
51-60	12	3	15	45.45
61-70	6	1	7	21.21
Above 70	4	1	5	15.15
	26	7	33	

The incidence of combined Ischemic Heart Disease & Systemic Hypertension is in the age group of 51-60 years (ie) 45.45% closely followed by 61-70 yrs(ie) 21.21%

Males have more incidence of IHD and SHD than Females.

CORRELATION BETWEEN DURATION OF DIABETES

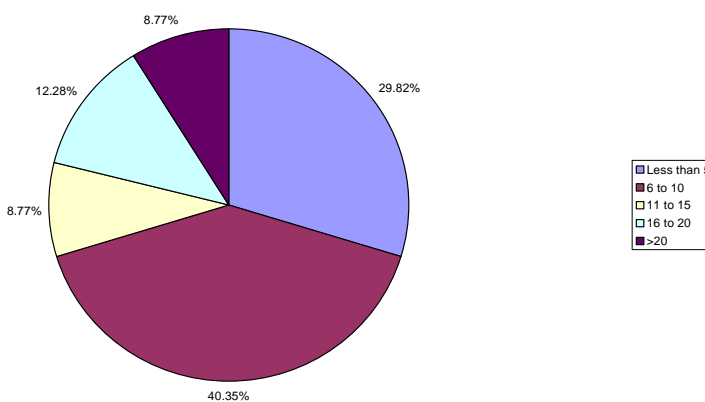
MELLITUS

AND INCIDENCE OF ISCHEMIC HEART DISEASE

S.No	Duration of Diabetes Mellitus In Years	No. of Cases of Ischemic Heart Disease	Percentage
1	Less than 5	17	29.82
2	6-10	23	40.35
3	11-15	5	8.77
4	16-20	7	12.28
5	>20	5	8.77

The incidence of IHD is maximum, 6 – 10 yrs after the onset of DM. It is about 40.35%

CORRELATION BETWEEN DURATION OF DM AND INCIDENCE OF IHD



Discussion

DISCUSSION

Cardiac involvement in diabetes commonly manifest as coronary artery disease (CAD), which alone accounts for the majority of mortality in diabetes.

a. Prevalence of IHD in Diabetes in our study is 54.3%, whereas in other studies are as follows.

Palhaniaand Sactar	-21.8%
Sterns ei.-tal	- 37.00%
Brady V Biyfoglee	- 40%
Lind back	- 63%
Several studies in India indicates	7-14%.

The discrepancy in the incidence of Ischemic Heart Disease among diabetes may be explained as follows :

(i) different criteria adopted among various authors to diagnose Ischemic Heart Disease, ii) Presence or absence of various associated risk factors such as hypertension, Hyperlipidemia, obesity, cigarette smoking, alcoholism and Type 'A', personality.

The high incidence of Ischemic Heart Disease in the present study may be attributed to fast changing lifestyle adopted by the local population and the prevalence of risk factors in the subjects analysed (i.e.) 62%, having one or the other combined risk factors, which plays an important role in paving for the cardio vascular complications. In our study very advanced diagnostic methods such as coronary angiography gives an overall prevalence of CAD as high as 54.3%.

The relative risk of myocardial infarction is 50% greater in diabetic men and 15% greater in diabetic women as substantiated by G.S. Sainnani & Rajesh Samani⁶⁵ which correlates with the present study incidence of infarction of 45.6%.

In most of the patients, ischemia and infarction co-exist, it is mainly due to the silent ischemia, pain of which is blunted in Diabetes Mellitus, but infarction some how brings the patient to hospital and the infarction is being diagnosed.

Silent ischemia is more common in Diabetes Mellitus. As per literature it is 60-70%. In the present study it is 30 %. Silent ischemia is more common in patients with family History of diabetes. Those who smoke cigarettes also develop silent ischemia. Myocardial infarction is

also presented with more of complications and peculiar presentations such as atypical character, location, acute breathlessness due to left ventricular failure and some others presented with arrhythmias or hypertension without chest pain. In the present study death due to myocardial infarction is more common in female patients than in male. The percentage is 23% in female and 15% in males It correlates with the Framingham heart study. Bundle Branch Block is very common presenting arrhythmia in this study group. There were 7 cases in women 3 cases in men.

b. Prevalence of Systemic Hypertension :

In our study prevalence of SHT is 21%. Prevalence of hypertension is 20% in U.S. urban population. R.Gupta epidemiological evolution and rise CAD in India - Published in South Asian .J. of Preventive Cardiology⁶⁶ 1997 reveals an urban incidence of Systemic Hypertension of 29.5%.

The incidence of raised blood pressure is generally correlated with obesity, sedentary life style as in advanced age of Type₂ Diabetes Mellitus. In the present study also, the incidence of SHT in Diabetes Mellitus is well correlated with obesity and the other risk factors. The incidence of hypertension in the age group of 51-60 yrs is 46.29%.

C. Association of Systemic Hypertension and Diabetes

Mellitus

An association between Systemic Hypertension and Ischemic Heart Disease is well established. It is related to the underlying development of atherosclerosis which is firmly associated with incidence of both systemic hypertension and ischemic heart disease.

In Framingham study, the risk of Ischemic Heart Disease especially myocardial infarction was found to be influenced by both systolic and diastolic pressure. Even elevated systolic pressure increases the risk in contrast with the views that diastolic pressure is more important as a risk factor for Ischemic Heart Disease.

d. Analysis of Age incidence:

Ageing plays important role in modifying the incidence of Ischemic Heart Disease & Systemic Hypertension in all, particularly in Type₂ Diabetes Mellitus. The prime pathogenesis behind development of Ischemic Heart Disease and SHT is atherosclerosis of vessels, which is more diffuse and prevalent in diabetes due to dyslipidemia. As the age advances, the incidence of atherosclerosis also progress leading on to the occlusion of blood vessels macrovascular lesion and increased incidence of Ischemic Heart Disease and Systemic Hypertension .

In the present study the incidence of IHD & Systemic Hypertension is more in the age group of 51-60 years of 33.33 % in the former and 47.27 % in the later. Malhotra et al observes the peak incidence of 36.3% in the sixth decade and Vishnava et al observed a peak incidence of 36.3% in the sixth decade⁶⁶.

E. Correlation between the duration of diabetes and ischaemic heart disease.

In the present study the incidence of ischaemic heart disease is more during the 6-10 years after development of Diabetes Mellitus - the percentage is 40.35%.

Many workers believe that the incidence of Ischemic Heart Disease is well correlated with the duration of the diabetes, [Bradly & Bryfogle], Abukhatave and Lewis did not observe any increase in the incidence of Ischemic Heart Disease in proportion to the increase in the duration of the diabetic status. Banerjee Patrak and Vaishnava et al observed an increasing trend of Ischemic Heart Disease incidence when the diabetes was of more than five years duration.

Conclusion

CONCLUSION

- Diabetes mellitus plays an important role in the development and progression of Ischemic Heart Disease.
- Ischemic Heart Disease is the most common cause of morbidity and mortality in diabetes patients.
- Next to IHD, Hypertension plays an important role in the progression of diabetic complications and mortality.
- Because of autonomic neuropathy, silent ischaemia and infarctions are more common in DM. This also accounts for the higher incidence of IHD mortality in DM.
- Various risk factors like smoking, alcoholism, hypertriglyceridemia and family History all accelerates cardiovascular complications.

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Appendix

Proforma

PROFORMA

NAME:

AGE:

SEX: M/F

I.P.NO:

DOA:

DOD:

C/O:

CHEST PAIN-TYPE, RADIATION

SWEATING

PALPITATION

SYNCOPE

DYSPNOEA

PND, ORTHOPNOEA

GIDDINESS

NAUSEA & VOMITING

HOPI:

RISK FACTORS:

DM/HT/SMOKING

DIET:

VEG/NONVEG/MIXED

FAMILY HISTORY:

IHD/OBESITY/DIABETES/HYPERTENSION/

HYPERLIPIDEMIA

TREATMENT HISTORY:

DRUG INTAKE

MARITAL HISTORY

GENERAL EXAMINATION:

- ❖ *OBESITY*
- ❖ *DYSPNOEA*
- ❖ *CYANOSIS*
- ❖ *CLUBBING*
- ❖ *PITTING PEDAL EDEMA*
- ❖ *XANTHALESMA*
- ❖ *TENDON XANTHOMAS*
- ❖ *ARCUS SENILIS*
- ❖ *PULSE*
- ❖ *BLOOD PRESSURE*

BP:

HEIGHT:

WEIGHT:

BMI:

CARDIOVASCULAR SYSTEM

APICAL IMPULSE *S1* *S2* *S3* *S4*

PERICARDIAL RUB

RESPIRATORY SYSEM:

ABDOMEN:

CENTRAL NERVOUS SYSTEM:

INVESTIGATION:

LIPID PROFILE:-

TOTAL CHOLESTEROL: mg%

TGL: mg% HDL: mg% LDL: mg%

ELECTROCARDIOGRAM:

ECHOCARDIOGRAM:

CHEST X-RAY

URINE ROUTINE

BLOOD SUGAR

BLOOD UREA

SERUM CREATININE

ABBREVIATIONS IN MASTER CHART

CM	- Cardiomegaly
HK	- Hypokinesia
DCM	- Dilated Cardiomyopathy
EF	- Ejection Fraction
LVH	- Left Ventricular Hypertrophy
TMT	- Tread Mill Test
MI	- Myocardial Infarction
N	- Normal
+	- Present
-	- Absent
M	- Male
F	- Female
T↓	- T wave Inversion